THE EPIDEMIOLOGY OF PSEUDORABIES

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The Epidemiology of Pseudorabies

Distribution and Incidence

A Calendar of Significant Events in the History of Pseudorabies.

1813	First reports of mad itch in cattle in the United States, (Tennessee and Ohio).
1823	Mad itch reported in cattle in Kentucky and Maryland--observation in Kentucky on relationship between the occurrence of the disease and feeding cattle cut corn with pigs in the same pen.
1889 & 1894	First reports of clinical evidence of pseudorabies in cattle in Europe, (Switzerland and Germany).
1902	First record of infectious nature of pseudorabies, by A. Aujeszky in Hungary, (from steer, dog, and cat).
1911	First report of pseudorabies in South America.
1914	First transmission of infection to pigs, by von Ratz.
1917	D. S. White in textbook of veterinary medicine considered mad itch synonymous with pseudorabies, but without experimental evidence.
1922	Manniger showed that pseudorabies virus infection caused clinical disease in swine, (Germany).
1931	Shope established that mad itch in American cattle was the same as Aujesky's pseudorabies. He also transmitted the infection to swine and observed mild clinical signs.
1932-1935	First reports of clinical disease in swine herds in Europe. Average mortality was 3-5%, sometimes as high as 10-30%, occasionally as high as 95% in suckling pigs.
1935	Shope reported on serological evidence of high prevalence of infection in swine from the middlewestern states, was still an unrecognized disease in pigs.

1937	First reports of pseudorabies in Africa.
1938	First reports of pseudorabies in the British Isles.
1943	First reports of clinical disease in suckling pigs in the United States.
1947	First reports of pseudorabies in China and Taiwan.
1962	First report of pseudorabies occurring as a serious disease affecting swine of all ages on several farms in Indiana.
1973	First report of pseudorabies in India.
1974	Reports of increasing incidence of virulent infections of swine in Indiana and Iowa.
1976	Pseudorabies in swine recognized as a national problem.
1977	Many states enact regulations to attempt to control the spread of pseudorabies. First pseudorabies vaccine licensed for use in the United States.

Laboratory Confirmations of Pseudorabies Virus Infections
(1974, 1975, 1976, and first six months of 1977)

		1974	1975	1976	(6 mo) 1977
1.	ALABAMA	1	0	5	1
2.	ARKANSAS	0	0	0	1
3.	CALIFORNIA	0	5	5	5
4.	CONNECTICUT	0	0	0	1
5.	FLORIDA	1	1	1	0
6.	GEORGIA	3	5	16	11
7.	ILLINOIS	3	8	75	46
8.	INDIANA	35	59	117	107
9.	IOWA	31	48	225	413
10.	KANSAS	15	20	30	9
11.	KENTUCKY	0	0	0	2
12.	MARYLAND	0	0	2	0
13.	MINNESOTA	0	4	11	26
14.	MISSISSIPPI	0	0	0	1
15.	MONTANA	0	0	1	1
16.	MISSOURI	1	10	20	25
17.	NEBRASKA	15	21	33	47
18.	NORTH CAROLINA	0	1	1	7
19.	OHIO	0	2	2	12
20.	OKLAHOMA	0	0	1	0
21.	PENNSYLVANIA	1	0	0	0
22.	SOUTH CAROLINA	0	0	1	1
23.	SOUTH DAKOTA	12	33	148(30)	5
24.	TENNESSEE	0	0	2	1
25.	TEXAS	1	7	13	5
26.	WISCONSIN	1	1	3	5
27.	WYOMING	0	0	3	0
	TOTALS	120	225	715	733

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Transmission of Infection

Pseudorabies virus (PrV) has been described as being a member of the herpesvirus group. This group consists of many members with common or similar chemical and physical properties. One of the common biological properties of the herpesviruses is their tendency to produce latent infections in their hosts. Pseudorabies virus is no exception. The occurrence of latent infections in swine which have recovered from natural infections is well documented. However, there still remain unanswered questions concerning the mechanisms of latent infections and initiation of virus shedding in convalescent swine. Additional studies are also needed to determine the incidence of latent carriers in the convalescent population. Although the figure, 3%, has been repeated by some individuals, no experimental data has been presented to support this conclusion. In a limited study, recently completed at Purdue University*, PrV was isolated from the tonsils of 13% (6 of 47) of a group of slaughter hogs which were known to have been infected when they were a few weeks old. In any case, latent carrier swine must be recognized as the major, if not only, reservoir of the virus and the most important vector--carrying virus from one area to another through normal industry movement of swine for breeding or finishing purposes.

Most herpesviruses have a very limited host range. Pseudorabies virus is unique among the group in this respect. It is the only known member of the group which is capable of infecting and causing disease in almost every species of warm-blooded animals. Only man and some of the higher apes seem to be refractive to infection. The epidemiology of Pr in species other than swine must be taken into consideration when attempting to control the spread of the disease.

It is generally accepted that PrV infections in all species except swine are uniformly fatal. The course of the disease in most animals is quite rapid. Death usually occurs within a few hours to 3 days after onset of clinical signs. Signs of disease may begin from 1 to 10 days after infection. The length of the incubation period is partially influenced by the infecting dose of virus.

* Kanitz, C. L., unpublished data.

In past studies, investigators have usually had little success with attempts to isolate virus from species other than swine. Consequently, all species except swine have commonly been referred to as dead-end hosts. The results of more recent studies, however, indicate that the dead-end host label is a misnomer. Virus has been found to be shed in saliva and nasal secretions of infected carnivores and omnivores. This excreted virus, as well as infected carcasses, can serve as sources of infection for other animals, negating the concept of dead-end hosts. In Indiana, during the last two years, there has also been clinical evidence of lateral transmission of PrV in ruminents, particularly in sheep where nearly entire flocks were lost without direct contact with swine.

Rather convincing circumstantial evidence has been accumulated to show that wildlife, particularly raccoons, may play an important role in spreading PrV from one farm to another within endemic areas. There have been many reports of diseased or dead raccoons on or near farms where swine were known to be infected with PrV. A number of these have been submitted to the diagnostic laboratory where PrV infection was confirmed. Recent studies at Purdue University have shown that raccoons are readily infected with PrV by consumption of infected baby pig carcasses or by being housed in contact with infected pigs where they ate and drank from common troughs. Likewise, susceptible pigs were infected by contact exposure to sick raccoons or by eating infected carcasses. During these studies virus was found to be present in the saliva of all infected raccoons tested and in nasal secretions of 90% of them. Although urine was not tested, 56% of the infected raccoons had virus present in their kidneys.

It is proposed that raccoons are infected with PrV when they visit infected swine herds to scavange improperly disposed of dead pigs or to eat at contaminated feeders. In some instances these infected animals may travel to nearby farms during the incubation period. Once clinical signs appear, their travels cease. If virus, shed in saliva or present in the carcass, is accessible to any pig on the farm, a new herd infection occurs. Raccoons have also been implicated in cases of Pr in cattle and goats. In these cases it

was thought that hay from mows where a number of raccoons were nesting was the source of infection. In one case hay, transported from a Pr endemic area to a Pr free area, was considered the only possible source of infection in a herd of goats.

In Indiana we have singled out raccoons as the species most probably involved as important infected vectors of PrV. This is compatible with the fact that there is a rather heavy population of this species in the Pr endemic area. Also, their natural habits and high susceptibility to infection make them ideal infected vectors within their range area. Other species may be more important in other areas of the country. The epidemiology of Pr in opposums is similar to that in raccoons. They are not as likely to be involved in the spread of the disease, however, since they do not coexist with man as well and are not found in such large numbers as raccoons. In areas where the terrain is suitable, muskrats may be potential vectors. There have been reports of observations of sick muskrats on swine farms prior to or during outbreaks of Pr in the herds. Unfortunately, laboratory confirmation of infection in these animals was not attempted. Studies at Purdue Unviersity have shown that muskrats, in contrast to common brown rats, are highly susceptible to infection with PrV by ingestion.

Carnivores such as dogs, cats, skunks and foxes are also readily infected by ingestion of improperly disposed of dead pigs or other infected carcasses. Their carcasses could, in turn, serve as sources of infection for susceptible swine herds if they were to die in the hogs lots or pastures.

The role of rodents in the epidemiology of Pr is somewhat controversial and needs further study. Most reports concern studies which were conducted using routes of inoculation other than oral and do not provide pertinent information concerning natural infections. Where rats from infected pig farms or orally infected rats have been studied the investigators concluded that: rats are relatively resistant to infection, infected rats do not excrete virus, and rats are not likely to be a reservoir for PrV or to play an important role in its spread. Mice have been shown to be more resistant to infection than rats. In spite of these findings, there seems to be a continuing, widely-held concept that rats are significantly involved in the epidemiology of Pr. Studies are presently being conducted at Purdue University to investigate the potential of rodents to be involved as vectors or reservoirs of PrV.

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Control Measures

Management Practices to Reduce Risks from Pseudorabies.

The most important means of spreading pseudorabies virus (PrV) is through movements of infected swine. Swine which have recovered from natural infections, but continue to carry and periodically shed infectious virus pose the greatest threat. Since these carrier swine cannot be identified by currently available methods, management and regulatory programs must be based on the assumption that all previously infected (serologically positive) pigs are possible carriers of PrV.

Some of the following suggested herd management practices may not always be applicable in light of current or future regulatory restrictions. Individual producers and their veterinary practitioners should evaluate the local situation in terms of type of production, risk of infection and effect of regulatory restrictions, and then devise a management program most suitable for the particular herd in question. In general, a choice must be made between two approaches to disease control--keep clean or keep immune.

The ideal way of dealing with any infectious disease is to keep it out of the herd. This may be difficult, if not impossible, when the herd is located in a Pr endemic area. However, a producer in a Pr free area should be able to maintain a clean herd by using protective management practices. The most important rule to follow is to not buy PrV when purchasing pigs. With or without mandatory regulations one should only purchase pigs which have been tested and found free of Pr antibody. All new additions to the herd (as well as any animals which have left the premises for any reason and are to be returned to the herd) should be held in isolation, completely separated from any contact with the herd, for at least 30 days. At the end of the isolation period they should be retested. If all animals in the group are still

serologically negative they may be added to the herd. If any are positive, either dispose of the entire group, or dispose of the reactors and submit the rest of the group to another cycle of isolation and retesting.

Many herds in endemic areas have become infected without bringing new animals into the herd and with no other known source of exposure. Most of these are presumed to have acquired the infection from wildlife vectors but this is difficult to confirm. Although one may not be able to prevent introduction of the virus into a herd in a high incidence area, some things can be done to decrease the chances of infection.

Efforts should be made to try to limit exposure of the herd to possible vectors. Limit pet population on the farm and keep dogs and cats out of the hog lots and houses if at all possible. Control rodent population and try to control other wildlife. Kill and remove any animals, particularly raccoons, that are observed to be sick or acting abnormal. Chill the carcasses immediately and submit them to a diagnostic laboratory for Pr testing. Also, restrict man and vehicle traffic from the hog lots and buildings. If the operation requires visitations by outsiders (particularly other swine producers or allied industry representatives who may visit several farms) provide them with clean overalls and boots before allowing contact with the herd.

Proper management can also lessen losses in the event PrV is introduced into the herd. Maintain separation of groups of pigs whenever possible. Don't mix and remake groups on a whim. Be convinced that it is necessary to do so. Have all disease conditions properly diagnosed. If Pr is suspected, submit specimens for laboratory confirmation immediately. Properly dispose of all dead pigs. Do not allow their carcasses to serve as a source of infection for other pigs in the herd or for vectors which may carry the virus to neighboring herds. Losses can be minimized if early diagnosis allows implementation of a vaccination program before the virulent virus spreads through the herd.

Vaccination Potential in Controlling Pseudorabies.

The recent licensing of a modified live virus vaccine* for the immunization of swine against Pr has made available a means by which high risk herds can be protected from the sometimes devastating losses resulting from an acute infection with virulent virus. When properly used the vaccine can also protect against the periodic losses that often occur in chronically infected herds and can be instrumental in eliminating virulent virus from such herds.

Proper use of Pr vaccine requires some understanding of what vaccination can and cannot accomplish. A single intramuscular inoculation with the licensed vaccine will effectively protect pigs of all ages against development of Pr when exposed to virulent virus. Although vaccination immunity does not prevent infection of the upper respiratory passages with the field virus, it does limit virus replication in these tissues and accelerates elimination of the virus from infected animals. Current data has not shown the establishment of latent carrier states in swine infected with virulent virus after vaccination. It must be pointed out, however, that vaccination of swine after they have been naturally infected with field virus will not eliminate carrier states already established.

The mechanism by which the vaccine protects pigs is not clear. The level of serum neutralizing antibody resulting from vaccination is not high enough to provide protection. Local cellular immunity or secretory antibody cannot be factors, since the vaccine virus is localized at the site of inoculation and is not found in the upper respiratory tract or the brain. The rapid, marked rise of neutralizing antibody after challenge of vaccinated animals suggests that the probable mechanism of protection involves stimulated or primed cellular elements in the blood. One point of practical importance involved here

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* PR-Vac, Norden Laboratories, Lincoln, Nebraska

is that it is unlikely that significant immunity will be conferred on pigs nursing vaccinated dams. Even pigs nursing sows with much higher antibody levels, resulting from natural infections, are poorly protected and will succumb to high challange doses of virulent virus. It is important, therefore, to vaccinate all newborn pigs as soon as maternal antibody levels have declined to a level where they will not interfere with the vaccine. Since this will vary from litter to litter and pig to pig it is best advised to vaccinate all pigs at 2 to 3 weeks of age and again at weaning.

Because of possible conflicts with regulatory programs it is recommended that Pr vaccine be used only in herds which are already infected, or are at a high risk of infection. In newly infected herds it would be desirable to immediately vaccinate all swine on the premises as soon as the disease is diagnosed. Although the vaccine will have little effect on the course of the disease in pigs already infected, overall losses may be considerably lessened if vaccination can be effected before the field virus has spread throughout the herd. A follow-up program, after the initial outbreak of the disease has run its course (and a program for managing a chronically infected herd), should be designed to maintain total herd immunity by continued vaccination, and systematically eliminate all animals which were on the premises at the time of the initial infection. All new additions should be vaccinated at least 21 days before they are brought into the herd and all newborn pigs should be vaccinated as previously described. Unless future duration of immunity studies indicate otherwise, breeding animals should be revaccinated before they are brought into service and twice yearly thereafter.

Using the above program, herd integrity can be maintained without the loss of valuable bloodlines while field virus is eliminated by removing those animals which are most likely to be latent carriers. All baby and growing pigs on the premises during the initial outbreak should be disposed of as soon as possible through feeder pig channels or to slaughter. Breeder replacements may be retained from pigs born after signs of disease are no longer present in the herd. Replacement of the breeding herd should then be accelerated in order to remove possible latent carriers as quickly as possible.

The decision to use Pr vaccine in a clean herd should be based on the potential risk of infection. Any herd located in an area where infected herds are known to exist is at risk, even though direct contact is not involved. The possible transmission of the virus from herd to herd by infected vectors poses a continual threat that is not easily controlled. Another threat to the susceptible herd is the proximity of pig finishing operations where pigs of unknown status are being shipped in. Such herds are often infected with PrV without visible evidence of the infection. In most instances these herds are maintained until slaughter without reason for laboratory confirmation of their infected status.

The type of vaccination program used to protect a clean, high risk herd may be dictated by the type of operation involved and the demands of regulatory restrictions. The totally immune herd concept would provide the greatest protection and is advised for the commercial, farrow to finish operation. The seedstock producer, however, may have to make use of a limited vaccination program if his market requires sales of serologically negative pigs. In this case he could partially protect himself against serious losses from a virulent infection by maintaining a vaccinated, immune breeding herd. However, he could not vaccinate progeny destined for sale. These unvaccinated offspring would be completely separated from the vaccinated herd at weaning and raised in isolation. After colostral antibody has dropped to an undetectable level they should be considered to be clean, serologically negative swine and be approved for sale for breeding purposes. It must be recognized that with this type of herd management, if infection with virulent PrV occurs, losses could be quite heavy among the unvaccinated young pigs.

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